myocardialization reflects the inability of B^C/B^C myocytes in the OFT to become polarized, disassemble their cell–cell adhesion complexes and migrate into the cardiac cushions. NM II is known to play an important role in each of these processes.

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Understanding the impact of γ-Secretase on cell death in *Drosophila*

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Aph-1 is a multi-pass transmembrane protein that works together with Presenilin (Psn), Nicastrin (Nct), and Pen-2, to form γ-Secretase, a protease that normally cleaves type I transmembrane proteins like Notch, cadherins and the Amyloid Precursor Protein (APP). Loss of function of any one of these four components of the γ-Secretase complex results in a failure of Notch signal transduction and improper cellular adhesion. We are interested to learn whether γ-Secretase regulates cell death. We found, during the wing development in *Drosophila*, that cells lacking Aph-1 activity seem prompt to cell death. A modified form of Psn protein, however, could rescue this phenomenon without rescuing Notch signal transduction. We are currently investigating whether γ-Secretase or Psn alone contributes to regulation of cell death during animal development in *Drosophila*.

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